Original Article

Qualitative and Quantitative Electrocardiogram Parameters in a Large Cohort of Children with Duchenne Muscle Dystrophy in Comparison with Age-Matched Healthy Subjects: A Study from South India

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Abstract

Background: Electrocardiography (ECG) remains an excellent screening tool for cardiac assessment in Duchenne muscular dystrophy (DMD), but an accurate interpretation requires comparison with age-matched healthy controls. **Objective:** We examined various ECG parameters in children with DMD, in comparison with age-matched controls. **Methods:** Standard 12-lead ECG tracings of serial patients were screened for quality and selected. Controls were healthy, age-matched school-going children. Both quantitative and qualitative ECG parameters were analyzed. **Results:** After screening, ECGs from 252 patients with DMD (8.32 ± 3.12 years, 2–21 years) and ECGs from 151 age-matched healthy controls (9.72 ± 2.23, 4–19 years) were included. A significantly higher heart rate, shorter R–R interval, and taller R wave in V1 were seen across all age group of DMD in comparison to controls, with the difference increasing with age. While QT prolongation was seen in all age groups of DMD, QTc prolongation was seen only at 10 years or more. Incomplete right bundle branch block (RBBB) and pathological Q waves in inferolateral leads were exclusive in DMD, with the latter declining with age. Evidence for left ventricular (LV) pathology, such as tall R in V5/V6, increase in SV1 + RV6 height, and QRS complex duration, were seen only in the age group of 10 years or more. **Conclusion:** Stratification based on age and comparison with age-matched healthy subjects showed that several ECG parameters were influenced by age, and it also identified age-dependent evidence for LV pathology and QTc prolongation in DMD.

Keywords: Cardiac screening, Duchenne muscular dystrophy, ECG, QT interval, tall R

Introduction

Duchenne muscular dystrophy (DMD) is the most common muscular dystrophy, manifesting as progressive skeletal and cardiac muscle damage with devastating consequences. DMD is caused due to the pathogenic variants in the dystrophin gene. The use of steroids, spinal stabilization surgeries, and advances in ventilatory care have improved survival in patients with DMD, and the predominant cause of mortality has shifted from respiratory to cardiac reasons.[1,2] Due to the limitations in physical activity, early symptoms of cardiac dysfunction could remain masked in these children, and hence, periodic screening is strongly recommended.[3] Electrocardiography (ECG) remains an excellent screening tool, given its widespread availability, low cost, ease of application, and interpretation, especially as the ECG changes often precede the echocardiographic (echo) changes.[4] In most cohorts of children with DMD, the common ECG findings include short PR interval and the electrographic evidence for a relative right ventricular hypertrophy (RVH)-like pattern, such as tall R wave in the right precordial leads, deep Q in the inferolateral leads, and prolonged QTc.[5-7] However, in view of the physiological maturation during pediatric age, many of these findings including short PR interval and RVH-like pattern can be found in normal children as well.[8-10] Ideally, to delineate ECG abnormalities, especially across different age groups of boys with DMD, comparison with age-matched controls is desirable. Besides, normative values for ECG parameters depend on the population being studied, and hence, distinguishing between pathological and nonpathological ECG

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changes may be difficult without age-matched healthy controls. Hence, through the current study, we examined in detail the various quantitative and qualitative ECG parameters in children with DMD in comparison with age-matched controls, which consisted of healthy, school-going children.

Materials and Methods

Standard 12-lead ECG tracings of serial patients with DMD who attended a neuromuscular clinic in a tertiary hospital in South India between 2015 and 2021 were screened retrospectively. The control population was selected from healthy, age-matched children through a school-based survey. Only boys with normal birth and development, having no history or symptoms of cardiac disease, and with normal clinical cardiac examination were included in the control group. For the 3-year and less than 3-year age group, healthy male siblings of patients attending the neurology clinics were selected after screening. Patients with any chest wall deformities or kyphoscoliosis as per chart review were excluded. ECG was recorded using BPL Cardiart 8108, a portable six-channel ECG recorder with real-time display of 12-lead ECG with a 24-bit A/D converter, 0.05–150 Hz bandwidth, and 1000 Hz sampling frequency. All ECGs were done by qualified technicians, with the patient in the supine position and after a minimum of 10 min of rest in a climatized room at the same time period of the day. The paper printouts of the tracings were collected, and each ECG feature was systematically analyzed by the investigator (MSG) blinded to the diagnosis. We grouped the cases and control subjects based on age into ≤6 years, 6–10 years, and ≥10 years, and further analyses were done both within and between these patient and control groups. The quantitative ECG parameters analyzed included heart rate, RR interval, P wave and QRS duration, PR, QT and corrected QT interval (QTc), R and S wave amplitudes in the precordial leads, and QRS axis. We also examined the qualitative measures, which included bundle branch blocks, fragmentation of QRS complex, presence and location of abnormal Q waves in the limb and chest leads. Statistical analysis was done using Statistical Package for the Social Sciences (SPSS) Version 26 (IBM, Armonk, NY, USA). The study was approved by the institutional ethics committee.

Results

Three hundred and fifty-one patients with DMD were identified and after excluding patients with kyphoscoliosis and ECGs with artifacts and suboptimal recordings, 252 ECGs were analyzed. The control population included 151 healthy, age-matched school-going children. The mean age was 8.32 ± 3.12 years (range 2–21 years) for patients with DMD and 9.72 ± 2.23 years (range 3–19 years) in the control population.

There was a significantly higher heart rate and corresponding smaller R–R interval in ECG among patients with DMD of all age groups compared to healthy boys, with the difference being more prominent in the older age groups. A significant shortening of *P* wave duration and prolongation of QRS duration were seen only in ≥10 years age group, while prolonged PR interval compared to control was found only in the extremes of age groups [Table 1].

Tall R wave in V1 chest lead was a prominent feature in the patient group. A tall R in lead V1 was seen in healthy subjects as well, but a significant difference between the groups was seen across all ages, with the difference increasing with age. The R/S ratio in V1 was also higher across all patient groups. The height of the R wave in V5/V6, a measure of left ventricular (LV) pathology, was significantly higher compared to healthy subjects from age 6 and above. There was no significant difference in V1 S wave height between case and control groups. When the heights of V5/V6 R wave and V1 S wave were added, there was no significant difference between cases and healthy boys at <10 years, but it was significant in ≥10 years group. Hence, it was noted that V5/V6 R wave height increases after 6 years and combined V5/V6 R + V1 S wave height increases after 10 years in DMD.

Although QT interval showed prolongation in all three age groups, when corrected for the heart rate, a prolonged QTc was noted only in ≥10 years of age. No difference was seen in QRS axis between patients and healthy subjects. Among patients, a significantly higher prevalence of incomplete right bundle branch block (RBBB) was seen across all age groups, but complete RBBB, notching in V1 S wave, and QRS complex in inferior leads (fragmented QRS) were similar to those in the control group [Table 2].

Abnormal Q wave was present almost exclusively among patients, with only five out of 152 controls having abnormal Q waves. A gradual decline in their prevalence was noted with increasing age. The most common location was in inferior + lateral leads (I, augmented vector Left [aVL], V4–V6, II, III, augmented vector Foot [aVF]), followed by lateral leads (I, aVL, V4–V6).

Table 3 summarizes the differences in various ECG parameters across the age groups.

Discussion

The distinctive ECG features in our large cohort of patients with DMD were that across all age groups, compared to healthy boys, the resting heart rate was higher and R–R intervals were shorter, abnormal Q waves were present in the inferolateral leads, tall R waves were seen in V1 lead with increased R/S ratio, and incomplete RBBB was present. Interestingly, evidence for LV pathology, such as tall R in V5/V6 leads and increase in SV1 + RV6 height characteristic in DMD, and prolonged QTc and QRS complex duration were seen only in the older age group. No difference in the QRS axis or QRS fragmentation was found in any of the age groups [Table 3].

The versatility of ECG as a screening tool in DMD is that besides being easily accessible and administrable, it is more sensitive than echo and even cardiac magnetic

Table 1: Comparison of the ECG quantitative variables among patients and controls

	Age group 1 (≤6 years)			Age group 2 (7–9 years)			Age group 3 (≥10 years)		
	Cases (*n*=67)	Controls (*n*=20)	*P*	Cases (*n*=112)	Controls (*n*=40)	*P*	Cases (*n*=73)	Controls (*n*=92)	*P*
Height V1 R wave	1.23±0.64	0.86±0.39	0.017	1.27±0.68	0.74±0.4	0.000	1.11±0.59	0.56±0.32	0.000
Height V5/V6 R wave	2.41±0.76	2.37±0.72	0.825	2.70±0.88	2.17±0.73	0.001	2.43±0.86	2.07±0.63	0.002
Height V1 S wave	1.06±0.51	1.08±0.45	0.866	1.15±0.58	1.33±0.52	0.082	1.10±0.54	1.12±0.42	0.753
V1 R: V1 S wave ratio	1.34±0.51	0.85±0.33	0.011	1.35±1.09	0.62±0.51	0.000	1.18±1.01	0.54±0.33	0.000
Height V5/V6 R + V1 S wave	3.47±1.02	3.45±0.92	0.934	3.86±1.15	3.5±0.88	0.082	3.53±1.14	3.19±0.84	0.031
Heart rate	112.04±17.97	99.4±11.66	0.004	104.10±12.47	91.68±10.78	0.000	97.74±13.11	84.43±13.24	0.000
R–R interval	548.34±83.1	610.89±65.66	0.003	584.36±68.21	663.80±82.56	0.000	624.77±83.49	728.02±114.88	0.000
P wave duration	84.08±7.48	86.95±8.17	0.144	83.98±10.07	84.33±8.00	0.846	84.65±8.82	90.21±7.82	0.000
PR interval	115.96±12.04	127.2±16.6	0.001	116.92±17.57	121.58±12.96	0.128	116.29±14.65	129.83±16.48	0.000
QRS duration	72.64±3.32	70.20±6.42	0.090	75.65±7.37	73.28 6.50	0.073	78.32±7.76	75.58±6.14	0.012
QT interval	305.85±23.12	318.25±17.98	0.031	318.26±21.35	336.78 20.10	0.000	329.25±22.17	347.47±21.34	0.000
Corrected QT interval (QTc – Bazett's)	415.04±18.86	408.95±16.68	0.197	417.72±17.52	414.53 13.73	0.298	418.38±16.00	409.80±17.06	0.001
QRS axis	62.72±23.82	74.2±26.11	0.068	67.68±26.12	64.58 32.37	0.546	66.93±39.09	67.16±29.56	0.965

ECG=Electrocardiography

Table 2: Comparison of the qualitative ECG variables between patients and controls

	Age group 1 (≤6 years) (*n*=87)			Age group 2 (7–9 years) (*n*=152)			Age group 3 (≥10 years) (*n*=165)		
	Cases (*n*=67)	Controls (*n*=20)	*P*	Cases (*n*=112)	Controls (*n*=40)	*P*	Cases (*n*=73)	Controls (*n*=92)	*P*
Complete RBBB *n* (%)	5 (7.5)	0 (0)	0.208	10 (8.9)	5 (12.5)	0.516	7 (9.6)	9 (9.8)	0.967
Incomplete RBBB *n* (%)	17 (25.4)	1 (5)	0.048	30 (26.8)	0 (0)	0.000	17 (23.3)	9 (9.8)	0.018
Notching V1 S wave *n* (%)	25 (37.3)	6 (30.0)	0.549	42 (37.5)	10 (25)	0.153	22 (30.1)	27 (29.3)	0.912
Notching in II, III, aVF *n* (%)	21 (31.3)	4 (20.0)	0.325	47 (42)	10 (25.0)	0.057	28 (38.4)	26 (28.3)	0.170
Abnormal Q waves *n* (%)	43 (64.2)	3 (15)	0.000	60 (53.6)	1 (2.5)	0.000	34 (46.6)	1 (1.1)	0.000
Q wave location – lateral leads *n* (%)	14 (20.9)	1 (5.0)	0.099	24 (21.4)	0 (0.0)	0.001	10 (13.7)	1 (1.1)	0.001
Q wave location – inferior leads *n* (%)	0 (0.0)	0 (0.0)	-	2 (1.8)	0 (0.0)	0.395	3 (4.1)	0 (0.0)	0.05
Q wave location – inferior and lateral leads *n* (%)	29 (43.3)	2 (10)	0.006	34 (30.4)	1 (2.5)	0.000	20 (27.4)	0 (0.0)	0.000

ECG=Electrocardiography, RBBB=Right bundle branch block

resonance imaging (MRI) in picking up early myocardial involvement.[11,12] Each of the ECG parameters represents unique electrophysiological events in the cardiac cycle and thus can reflect in advance the evolving pathological changes in DMD-related cardiomyopathy [Supplementary Table 1]. There is an evolution in ECG findings from infancy to adolescence, which reflects changes in cardiac physiology. At birth, the amplitude of R waves due to relative RVH will be higher in the right precordial leads, and this would normally reverse with age. However, R/S may remain more than 1 even up to 8–12 years.[10,13] This would progressively reverse with age in normal children.[13] The inverse is true for S wave amplitude. The R/S ratio in V1 may remain more than 1 even up to 8–12 years.[10] Thus, an increased R/S ratio in lead V1 is a sign of pathology only when compared to age-matched controls and can be seen in a variety of conditions including DMD. This highlights the relevance of age, but existing data differs in the relationship between age and ECG changes in DMD.[6,8]

Dystrophin in the cardiac myocytes causes stress-related apoptosis, fibrofatty replacement, and scarring, which gradually extend from the epicardium to the endocardium and from the inferobasal region down toward the apex, resulting in thinner LV and eventually a dilated cardiomyopathy.[14-16] As the QRS complex comprises both right ventricular and LV vectors, the deficiency in the LV mass may result in an apparent increase in right ventricular depolarization, reflected as tall R wave height in the right precordial leads and deep Q waves in the inferolateral leads (pseudoinfarction pattern).[8] The associated right ventricular conduction delay results in an incomplete RBBB. But the exact cause for these patterns in DMD remains debatable, and the cardiac involvement is found to be unrelated to the type or location of the variant.[17]

One of the characteristic findings which became more apparent with age was the elevated resting heart rate and the shortened PR interval, the latter, in particular, representing sympathetic overactivity. Sympathetic overactivity revealed an association with underlying myocardial dysfunction. Besides an increase in heart rate, a decrease in heart rate variability signifying an autonomic imbalance was also a marker of myocardial fibrosis, even when the cardiac ejection fraction was normal.[18] This

Table 3: Summary of the comparison of ECG changes between patients and healthy controls

Findings seen across all age groups	
Quantitative	**Qualitative**
• Height in V1 R • R/S in V1 • Heart rate and RR interval • QT interval • Sinus tachycardia	• Incomplete RBBB • Abnormal Q waves in inferolateral leads
Findings seen in 6 years or older age group	
Quantitative	**Qualitative**
• Abnormal height of V5/6 R wave	• Q waves in lateral leads alone
Findings seen in 10 years or more age group	
Quantitative	**Qualitative**
• Short P wave duration • Abnormal SV1 + RV6 • Prolonged QTc • Prolonged QRS duration	
Findings not different compared to controls	
Quantitative	**Qualitative**
• S V1 height • QRS axis	• Notching of QRS complex in V1 and II, III, aVF • Q wave in inferior leads

Significantly shorter PR interval was noted in age groups of <6 years and >10 years. ECG=Electrocardiography, RBBB=Right bundle branch block

highlights the significance of cardiac autonomic dysfunction in children with DMD, which could prove to be an important biomarker for early detection of an actionable cardiac involvement. Despite the influence of age in PR interval, wherein it lengthened with age, a significant difference was found in all patient groups compared to controls, except in the 7–9-year age group, where it did not reach statistical significance.

There are some discordances in terms of the relationship between age and the other ECG changes in DMD in comparison to some previous studies. Among 78 steroid-naïve patients in <6 years age group, ECG changes such as sinus tachycardia, short PR interval, and prolonged heart rate-corrected QT interval were not common, while changes in LV pathology predominated.[12] Another study, which compared between the abnormal and normal ECG parameters in patients with DMD, found only ventricular repolarization to be different and that it was encountered more commonly in younger age. QT interval, corrected for the heart rate, is a marker of ventricular depolarization and has been proposed as one of the earliest myocardial changes in DMD.[19] Our data showed that compared to healthy controls, prolonged QTc was seen mainly in the older age group. Among the other progressive ECG changes, PR and QRS interval prolongation was noted, with the latter being a potential risk factor for Atrioventricular (AV) dissociation.[20] Although a number of cardiac arrhythmias including atrial and ventricular tachyarrhythmias are described in DMD, none of our patients were noted to have any rhythm abnormalities, except sinus tachycardia, likely because of the brevity of the conventional ECG recording.[15]

Notable too were the abnormal R height in V5/6 and the elevated SV1 + RV6 height in patients with DMD. These changes in the lateral precordial leads are a marker of LV hypertrophy and have only rarely been reported in DMD. To our knowledge, only one study has noted ECG markers of LV pathology to be the most common finding in their cohort of less than 6-year-old patients with DMD, which included tall R in V1 and elevated R/S ratio in V6.[12] But in our cohort, these changes were seen only among older age groups. The exact mechanism for these changes is difficult to explain, and no correlation has been found between these ECG changes and cardiac structural changes as detected by echo.[5,19,21] It is possible that the progression in pathology and deviation from the normal electrophysiological changes mean that certain differences become more apparent only when compared with age-matched controls.[8]

Our study has a few limitations. For the control population, we selected apparently healthy school-going children based on history and clinical examination, but an echo was not done to exclude a preexisting heart disease. Use of Holter monitoring would have enabled to identify cardiac arrythmias, which were hardly seen in our cohort of patients.[22,23] The inclusion of clinical correlate, echo, or cardiac MRI parameters would have given more information and made the study more robust. Data regarding serial ECG changes in the same cohort could have been more illuminating on the age-dependent evolution. We have not attempted genotypic or phenotypic correlation with ECG data, although no such correlations have been found in previous studies.

Conclusion

Stratification based on age and comparison with age-matched healthy subjects provided unique insights into the ECG changes among children with DMD. The current study noted that several ECG parameters were influenced by age and identified age-dependent evidence for LV pathology and QTc prolongation, both of which were prominent in the older age group. Knowledge about these various age-dependent changes can expand our repertoire in screening patients with DMD for cardiac involvement to start timely treatment.

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Conflicts of interest

There are no conflicts of interest.

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Supplementary Table 1: ECG parameters and their physiological correlation

Parameter	Electrophysiology
P wave	Atrial depolarization; spread of depolarization from SA node to right and left atrial myocardium
PR interval	Time between onset of atrial and ventricular depolarization
QRS complex	Ventricular depolarization; first phase depolarization of the interventricular septum from the left to the right and second phase simultaneous depolarization of the right and left ventricles
QT interval	Total duration of ventricular depolarization and repolarization
T wave	Ventricular repolarization; from ventricular epicardium to endocardium

Reference[20]